Published in final edited form as:
Cancer Epidemiol. 2013 February ; 37(1): 5–10. doi:10.1016/j.canep.2012.09.002.

Esophageal cancer incidence by histological type and overall: Puerto Rico versus the United States SEER population, 1992–2005

Lorena González[a], **Priscilla Magno**[b,c], **Ana P. Ortiz**[c,d], **Karen Ortiz-Ortiz**[e], **Kenneth Hess**[f], **Graciela M. Nogueras-González**[f], and **Erick Suárez**[a,d,*]

Lorena González: lorena.gonzalez2@upr.edu; Priscilla Magno: priscilla.magno@upr.edu; Ana P. Ortiz: ana.ortiz7@upr.edu; Karen Ortiz-Ortiz: kortiz@rcpr.org; Kenneth Hess: khess@mdanderson.org; Graciela M. Nogueras-González: gnoguera@mdanderson.org

[a]University of Puerto Rico/The University of Texas MD Anderson Cancer Center Partnership in Excellence in Cancer Research Program, School of Medicine, Medical Sciences Campus, University of Puerto Rico, San Juan, PR

[b]School of Medicine, Medical Sciences Campus, University of Puerto Rico, San Juan, PR

[c]Cancer Control and Population Sciences Program, University of Puerto Rico Comprehensive Cancer Center, San Juan, PR

[d]Department of Biostatistics and Epidemiology, Graduate School of Public Health, Medical Sciences Campus, University of Puerto Rico, San Juan, PR

[e]Puerto Rico Central Cancer Registry, University of Puerto Rico Comprehensive Cancer Center, San Juan, PR

[f]Department of Biostatistics, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

Abstract

Objective—The aim of our study was to compare the age-standardized incidence of esophageal cancer (EC) in Puerto Ricans (PRs) with that for non-Hispanic White (NHW), non-Hispanic Black (NHB), and Hispanic (USH) groups in the United States (US) as reported by the Surveillance, Epidemiology, and End Results program for the 1992–2005 period.

Methods—We computed age-standardized and age-specific incidence (per 100,000 individuals) of EC during 1992 to 2005 using the World Standard Population as reference. The percent changes for age-standardized incidences, from 1992–1996 to 2001–2005, were calculated. The relative risks (RR) and the standardized rate ratios (SRR) were estimated, along with 95% confidence intervals (CI).

Results—Age-standardized rates (ASR) of adenocarcinomas (AC) showed increases for most racial/ethnic groups from 1992–1996 to 2001–2005. All racial/ethnic groups showed ASR

*Corresponding author: Erick Suárez, PhD, University of Puerto Rico, Medical Sciences Campus, Department of Biostatistics and Epidemiology, PO Box 365067, San Juan, PR, 00936-5067. Phone (787) 758-2525 x-1430; Fax (787) 764-5831; erick.suarez@upr.edu.

Conflict of interest: None declared

reductions for squamous cell carcinomas (SCC). For both sexes, PRs had lower AC incidences than NHW and USH but higher than NHB. For those younger than 80 years of age, PR men showed higher SCC incidences than NHW but lower than NHB ($P < 0.05$). The incidence of SCC was about two times higher in PR men than USH men (SRR: 2.16; 95% CI = 1.65–2.88). Among women, the RR for SCC increased with age when comparing PRs to groups in the US.

Conclusion—Incidence disparities were observed between PRs and other racial/ethnic groups in the US. These differences and trends may reflect lifestyles of each racial/ethnic group. Further studies are warranted to explain these disparities.

Keywords

Esophageal cancer; Incidence; Puerto Rico; Relative risks; Standardized rate ratios

1. Introduction

Esophageal cancer (EC) has been ranked as the 8th most common cancer and the 6th most common cause of cancer death in the world.[1] The American Cancer Society estimated a total of 16,980 new cases and 14,710 deaths from EC for the year 2011 in the United States of America (US).[2] Nonetheless, countries like Iran, India, South Africa, and north China present rates that are 10 to 100 times higher than the US.[2] Also, relative to the World Standard Population, the Puerto Rican (PR) population has higher age-standardized incidence per 100,000 individuals than the US, in both men (7.3 vs. 5.8) and women (1.9 vs. 1.2).[1]

In the US, different incidence and mortality trends have been seen between racial and ethnic groups. The US Hispanic (USH) population, for example, has lower EC incidence than the non-Hispanic white (NHW) and non-Hispanic black (NHB) populations; the latter group having the highest rates.[3,4] In addition, men clearly present a higher risk of developing EC than women.[3,5,6] Distinct geographic, racial, and sex differences play an important role in EC trends.

During the past several decades, the sudden increase in EC incidence has become a major concern in healthcare. This increase has been explained by the rise of esophageal adenocarcinoma (AC) [7], which is one of the main histology types of EC along with squamous cell carcinoma (SCC). During the early 1980s, AC accounted for <15% of all EC whereas now it represents >60% [5], displacing SCC and becoming the most common histological type of EC in the US and western Europe.[7,8] The incidence of SCC has decreased by 3.6% per year between 1998 and 2003 while that of AC increased 2.1%.[6] In the US, most of the increase in AC has occurred in NHW of both sexes.[9,10] Nonetheless, SCC remains 6 times more likely to occur in black men than in white men.[11]

The comparison of PR cancer statistics with those of racial/ethnic groups in the US is of interest because of the sociocultural relationship between Puerto Rico and the US.[12] Such a comparison is not only necessary to understand differences and similarities in EC occurrence but could also provide relevant information on the influence of different factors on disease occurrence. We know of no comparison between PRs and US groups for a recent period or for the different histological types of EC. The aim of our study was to compare the age-standardized incidence of EC in PRs with that for USH, NHW, and NHB groups in the US as reported by the Surveillance, Epidemiology, and End Results (SEER) program for the period 1992–2005.

2. Methods

2.1. Data Sources

As described in other studies [13,14], data sources for this analysis included the SEER program and the Puerto Rico Cancer Center Registry (PRCCR). The PRCCR, part of the National Program of Cancer Registries, is administered by the Centers for Disease Control and Prevention (CDC) and uses the coding standards of the SEER program and of the North American Association of Central Cancer Registries, which makes the registry's data fully comparable to the SEER data. The criteria specified in the third revision of the International Classification of Diseases for Oncology (ICD-O-3) were used to select all cases of EC from 2001 and later (site codes C150–C155 and C158-159) for this analysis.[15] Cases from 1992 to 2000 were originally reported using ICD-O-2 and later converted to ICD-O-3 by the SEER program.[16] Esophageal cancer incidences data from 1992 to 2005 for PRs were obtained from the PRCCR. Esophageal cancer incidences cases for 1992 to 2005 for each racial/ethnic group (NHW, NHB, and USH) in the US assessed in this study were obtained with the SEER*Stat 6.3.5 software (National Cancer Institute Surveillance Program, Bethesda, MD) and were based on SEER 13 registries database which includes the following cities and states: Atlanta (Georgia), Connecticut, Detroit (Michigan), Hawaii, Iowa, New Mexico, San Francisco-Oakland (California), Seattle-Puget Sound (Washington), Utah, Los Angeles (California), San Jose-Monterey (California), Rural Georgia, and the Alaska Tumor Registry. Hispanic ethnicity was identified by the SEER program using a combination of medical record review and matching surnames against a list of Hispanic surnames.[17] This study does not account for racial differences within the USH population.

2.2 Statistical Analysis

For each racial/ethnic group, we applied the direct method to compute EC age-standardized incidence (per 100,000 individuals) during 1992 to 2005 using the World Standard Population as reference.[18] To assess the trend of EC risk by sex from 1992–1996 to 2001–2005 period, we calculated the annual age-standardized rates [ASR (World)] for each period (1992–1996,1997–2000, and 2001–2005), as follows:

$$ASR(World)_i^k = \sum_{j=1}^{4} w_j \frac{d_{ij}^k}{n_{ij}^k}$$

where j represents a given age group, i represents a given ethnic group, k represents a given period, w is the proportion of people in the world population to be evaluated, d is the number of new cases or deaths, and n is the total population. The change in the ASR from the earliest and the latest study period (1992–1996 and 2001–2005) was calculated as a percentage as follows:

$$\%\text{change} = \frac{(Rate_{2001-2005} - Rate_{1992-1996})}{Rate_{1992-1996}} * 100$$

The significance of the percentage of change was determined by 95% confidence intervals (CIs) using the formulas from the US Census Bureau.[19] If zero was not included in this interval, significance was set at a P-value less than 5%.

To assess racial/ethnic group differences, we grouped the ASR(World) values during the study period (2001–2005) as follows:

$$ASR(World)_i = \sum_{j=1}^{4} w_j \frac{\sum_{k=2001}^{2005} d_{ij}^k}{\sum_{k=2001}^{2005} n_{ij}^k}$$

Then, the ratio of two standardized rates [$ASR\,(World)group_i / ASR\,(World)group_j$] between any two groups (i and j) was estimated with 95% CIs [20] to assess differences in EC incidence between the PR group and USH, NHB, and NHW groups. This ratio is referred to as the standardized rate ratio (SRR).

In addition, age-specific incidence (per 100,000 individuals) for different age groups was computed by sex for 2001–2005. On the basis of these rates, the relative risks (RR) were estimated with 95% CIs to determine relative differences among the study groups by sex and 10-year age group (50–59, 60–69, 70–79, and ≥ 80) using the Poisson regression model.[21] The interaction between age groups and racial/ethnic groups was also assessed using the likelihood ratio test. The reference groups in the age-specific RR estimation were NHW, NHB, and USH, each stratified by sex. The regression was performed using Stata/SE version 11.0 statistical software (Stata Corp., LP., College Station, TX).

3. Results

3.1 Trends of ASR(World)

All EC—Among men, NHW and USH showed an ASR(World) increase, from 1992–1996 to 2001–2005, but it was only significant for NHW (% change = 18.2; $P < 0.05$). On the other hand, both NHB men and PR men had significant reductions over time ($P < 0.05$; Table 1). Among women, the only observed increase was not statistically significant and occurred in NHW (% change = 1.1; $P > 0.05$). In contrast, PR, USH, and NHB women all had ASR(World) that decreased from 1992–1996 to 2001–2005. Only PR and NHB women had a significant decline (% change in PR = −44.8 and % change in NHB= −20.3; $P < 0.05$ for both).

AC and SCC—Age-standardized rates of AC showed increases for most racial/ethnic groups from 1992–1996 to 2001–2005 (Table 1). Despite the increase of AC in most of the groups, significant changes were only seen in NHW (% change in men = 44.6 and % change in women = 45.8; $P < 0.05$ for both sexes). Moreover, an AC reduction was observed among NHB men (% change = −12.5); however, this reduction was not significant ($P > 0.05$). Conversely, all racial/ethnic groups showed ASR reductions in squamous cell carcinoma from 1992–1996 to 2001–2005 (Table 1). The greatest incidence reductions for SCC were observed for PRs (% change in men = −44.2 and % change in women = −48.7; $P < 0.05$ for both sexes).

3.2 SRR and RR (2001–2005 period)

All EC—An age and racial/ethnic group interaction was observed when comparing EC incidence of PRs to those of NHB and NHW; a lower incidence for PRs was observed among persons between 50 and 79 years of age (Table 2). On the contrary, the oldest age group (≥80 years) showed consistently higher incidence for PRs than for NHB and NHW; it was only significant for the comparison between PR and NHB men (Table 2). For both sexes, PRs had higher incidences of all EC than USH did (SRR_{men}: 1.40, 95% CI = 1.16–1.69; and SRR_{women}: 1.88, 95% CI = 1.37–2.63) with no evidence of age and racial/ethnic group interaction ($P > 0.05$).

AC and SCC—None of the comparisons of AC incidence between PRs and the other racial/ethnic groups showed evidence of an interaction between age and racial/ethnic group ($P > 0.05$) (Table 3). For both sexes, PR had lower incidences of AC than NHW and USH but higher AC incidences than NHB (Table 3). Puerto Rican men had about two times the incidence of AC that NHB men did (SSR: 2.32, 95% CI = 1.51, 3.71).

Conversely to AC, for SCC, we found an interaction between age and racial/ethnic group when comparing incidences in PR women with those of NHW, NHB, and USH women. Among women, the RR for SCC increased with age (Table 4). Among men, PRs showed higher SCC incidences than NHW but lower than NHB for people younger than 80 years of age ($P < 0.05$; Table 4). The incidence of SCC was about two times higher in PR men than USH men (SRR: 2.16; 95% CI = 1.65–2.88).

4. Discussion

The results of this study indicate that EC incidences in PRs, depending on the EC histological type, differ from those in other racial/ethnic groups in the US. When histological types were evaluated separately, NHW showed the highest incidence of AC, whereas NHB showed the highest incidence of SCC for most of the periods studied. We found the incidence of AC was lower in PRs than in NHW and USH but higher than in NHB, whereas the incidence trends of SCC were generally opposite those of AC. As previously seen in other studies, the incidence of EC, both in PRs and the other racial/ethnic groups, was higher among men than women.[3,5,6,12,22] Partly, incidence differences between the sexes could be explained by differences in alcohol consumption and tobacco use [23,24], which have been strongly associated with SCC.[25,26]

Despite the elevated incidence of all EC in PRs, the island showed one of the largest declines in SCC incidence between 1992–1996 and 2001–2005 (Table 1). Behavioral Risk Factor Surveillance System (BRFSS) data [27] might explain why the SCC incidence has decreased in Puerto Rico and the US: from 1997 to 2010, the use of cigarettes decreased in both regions (14.4% to 11.9%in PRs and 23.2% to 17.3% in the US). Puerto Ricans had a lower prevalence of current smokers than the US during this period, but at the same time, the NHB population in the US had the highest prevalence [27], potentially explaining why the PRs showed a lower incidence of SCC than NHB in the US ($P < 0.05$) for both sexes. Despite a lower prevalence of current smokers, the PR population doubled and tripled the risk ($P < 0.05$) for SCC as compared to NHW and USH population, which could be explained by the duration of tobacco consumption rather than the number of cigarettes smoked by current smokers.[23,28] In addition, the prevalence of heavy drinkers also decreased slightly in Puerto Rico (3.8% to 3.0%) and the US (5.1% to 5.0%) from 2001 to 2010; NHW consistently showed the highest prevalence of drinking during this period, whereas the lowest prevalence has been frequently observed in NHB.[27] Furthermore, the risk of SCC does not seem to depend on the duration of alcohol consumption but on the mean daily intake [23,28], which could influence our results. The period effects for both factors (alcohol consumption and smoking) must also be considered.

One factor that has been continuously related to a reduction in the risk of SCC is a diet high in fiber and vitamins.[29,30] One of the major sources for fiber and vitamins is fruits and vegetables. From 1996–2009, the prevalence of persons in Puerto Rico consuming more than 5 servings of fruits and/or vegetables per day was 7.2% in 2000 and 17.7% in 2009.[27] In the US, the overall prevalence of consuming fruits two or more times per day decrease significantly from 34.4% to 32.4% whereas no significant change was observed for consuming vegetables three or more times per day (26.7% in 2000 and 26.3% in 2009).[31] Although our study design cannot make any assessment of biological relevance of fruits and

vegetables consumption on the development of EC these estimates could support the hypothesis that differences in EC between PR and other US racial/ethnic groups exist.

In opposition to the trends seen in SCC, PR and the other racial/ethnic groups in the US, except NHB men and PR women, showed an increased incidence of AC in the time periods from 1992–1996 to 2001–2005. The rise of AC could be due to an elevated exposure to risk factors such as obesity, gastroesophageal reflux disease (GERD), and Barrett's esophagus (BE), which have been consistently related to this histological type. For example, it has been suggested that obesity trends are temporally consistent with the epidemiology of AC.[32] Cossrow and Falkner [33] stated that the biggest rise in obesity rates was observed among NHB women, followed by USH and NHW women. This could explain why the largest change in AC incidence was observed for NHB women during the 1992–2005 period (% change = 85.7), although not significant.

Obesity is associated with about twice the risk of AC (OR: 2.2, 95%CI: 1.1–4.3) as compared to a normal body mass index (BMI).[34] Data from the BRFSS showed that 62.4% of NHW, 72.3% of NHB, and 65.4% of USH were overweight or obese in 2007.[27] Likewise, Pérez et al [35] found that 77.5% of the PR population (n=859) were overweight or obese and 49.0% showed excess of abdominal fat. Although the rate of obesity seems to be higher in the PR population than in the US population, an increased risk of esophageal AC was only observed when comparing PRs to NHB. However, excessive visceral adipose tissue (VAT) instead of BMI has been considered the main factor associated with developing AC among obese people.[36] Researches have found that larger VAT levels are more likely related to European genetic admixtures than to African genetic admixtures ($P < 0.013$ and $P < 0.001$, respectively) [37], which could be responsible for the higher risks of AC in the PR population than NHB but lower than NHW and USH. Furthermore, men have greater propensity than women to accumulate excess fat within the abdominal cavity despite the fact that women as a group tend to be more obese than men, which could be another explanation for incidence differences between the sexes.[38]

The effect of obesity, as well as GERD, may be a critical factor in the increased incidence of AC observed in this study. The prevalence of GERD in the US has been estimated to be 25%–35%.[39] According to the American Society for Gastrointestinal Endoscopy [40], 10 to 15% of patients with GERD develop BE and consequently are predisposed to AC. Data on GERD's prevalence in PRs have not been published; a questionnaire is in the process of being validated for this purpose.[41] Nevertheless, Altman et al. [42] have indicated that an increase in the proportion of US primary care visits for GERD occurred between the periods of 1990–1993 and 1998–2001 making this a reasonable explanation for an increase in AC incidence. However, BE could also be playing an important role in AC incidence rates and trends differences. Barrett's esophagus, which is a strong risk factor for AC and is a complication of chronic GERD, has shown significant differences by racial/ethnic group and sex in the US.[43,44]

4.1 Strengths and limitations

This study provided updated population-based data on EC, by histological type and overall, in PRs compared to racial/ethnic groups in the US. According to a CDC audit in the year 2003, 95.3% of all cancer cases diagnosed or treated in hospital facilities in Puerto Rico were appropriately reported to the PRCCR, which is a rate comparable to the US median (95%). However, some limitations of this study should be acknowledged. First, we were unable to collect data on individual risk factors because neither the PRCCR nor the SEER 13 program collects this information. However, our results suggest differences in rates and trends which may reflect different lifestyles across races and ethnicities. Second, our results may have been influenced by poor accuracy in the classification of Hispanic cancer cases in

the SEER 13 program. Nevertheless, results from a previous study showed that bias, when classifying cancer cases as USH, can be reduced by combining surname and medical record information.[45] Since this method has been used by the SEER 13 program when classifying persons as USH we do not expect major changes in our conclusions.

5. Conclusions

Despite the reduction of SCC incidences in PRs and all the racial/ethnic groups in the US ($P < 0.05$), data in this study show that SCC was still more common than AC among PR and NHB for each period (1992–1996, 1997–2000, and 2001–2005; Table 1). This result suggests it is necessary to continue addressing the substantial burden of SCC in these populations. Although there has been a great reduction in tobacco use, it is crucial to continue promoting and strengthening a healthy lifestyle including healthy dietary habits, reduced alcohol consumption, and reduced tobacco use. According to Doll and Peto [46], populations can achieve an 86% reduction in the risk of aerodigestive tract cancers by avoiding tobacco and alcohol use and increasing fruit and vegetable consumption; a 20% reduction in risk can be attributed to dietary change alone. Improving dietary habits can not only serve as a protective factor for SCC, as suggested by others researchers [47–50], but could also indirectly help reduce AC incidence because majorrisk factors for this histology are related to diet.[51]

In conclusion, incidence disparities were observed between PRs and other racial/ethnic groups in the US. As is seen for other types of cancer, these differences and trends may reflect lifestyles of each racial/ethnic group. Further studies are warranted to explain the disparities in EC incidence by racial/ethnic group.

Acknowledgments

We would like to thanks the PRCCR for providing the data for this study. We are also grateful to Zachary Bohannan and Kathryn Carnes, from MD Anderson Cancer Center, for editing and reviewing this document. Furthermore, we appreciate the support provided by the UPR/MDACC Partnership for Excellence in Cancer Research, National Cancer Institute (U54CA96297 and U54CA96300); the National Institute of Health-National Center for Research Resources, Research Centers for Minority Institutions (P20RR11126 and G12RR03051); the PR Clinical and Translational Research Consortium (1U54RR026139-01A1); the Case Comprehensive Cancer Center Training in Computational Genomic Epidemiology of Cancer (5R25CA094186-08); and the National Program of Cancer Registries (NPCR) of the CDC by the PRCCR (1U58DP000782-04). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Cancer Institute.

References

1. Ferlay, J.; Shin, HR.; Bray, F.; Forman, D.; Mathers, C.; Parkin, DM. GLOBOCAN 2008 v1.2, Cancer Incidence and Mortality Worldwide: IARC CancerBase No. 10 [Internet]. Lyon, France: International Agency for Research on Cancer; 2010. Available from: http://globocan.iarc.fr
2. American Cancer Society. Esophageal cancer [Internet]. Atlanta, Georgia: American Cancer Society Inc; Available from: http://www.cancer.org/Search/index?QueryText=dysphagia
3. Phillips AA, Jacobson JS, Magai C, Consedine N, Horowicz-Mehler NC, Neugut AI. Cancer incidence and mortality in the Caribbean. Cancer Invest. 2007; 25(6):476–83. [PubMed: 17882661]
4. Carozza SE, Howe HL. Patterns of cancer incidence among US Hispanics/Latinos, 1995–2000. Cancer Causes Control. 2006; 17(8):1067–75. [PubMed: 16933057]
5. Baquet CR, Commiskey P, Mack K, Meltzer S, Mishra SI. Esophageal cancer epidemiology in blacks and whites: racial and gender disparities in incidence, mortality, survival rates and histology. J Natl Med Assoc. 2005; 97(11):1471–8. [PubMed: 16334494]
6. Trivers KF, Sabatino SA, Stewart SL. Trends in esophageal cancer incidence by histology, United States, 1998–2003. Int J Cancer. 2008; 123(6):1422–8. [PubMed: 18546259]

7. Blot WJ, McLaughlin JK. The changing epidemiology of esophageal cancer. Semin Oncol. 1999; 26(5 suppl 15):2–8. [PubMed: 10566604]
8. Layke JC, Lopez PP. Esophageal cancer: a review and update. Am Fam Physician. 2006; 73(12): 2187–94. [PubMed: 16836035]
9. Vega KJ, Jamal MM, Wiggins CL. Changing pattern of esophageal cancer incidence in New Mexico: a 30-year evaluation. Dig Dis Sci. 2010; 55(6):1622–1626. [PubMed: 19688596]
10. Cook MB, Chow WH, Devesa SS. Oesophageal cancer incidence in the United States by race, sex, and histologic type, 1977–2005. Br J Cancer. 2009; 101(5):855–859. [PubMed: 19672254]
11. Erickson KL. Dietary pattern analysis a different approach to analyzing an old problem, cancer of the esophagus and stomach. Am J Clin Nutr. 2002; 75(1):5–7. [PubMed: 11756053]
12. Martínez I, Torres R, Frías Z. Cancer incidence in the United States and Puerto Rico. Cancer Res. 1975; 35(11 Pt 2):3265–71. [PubMed: 1238165]
13. Suárez E, Calo WA, Hernández EY, Díaz EC, Figueroa NR, Ortiz AP. Age-standardized incidence and mortality rates of oral and pharyngeal cancer in Puerto Rico and among non-Hispanics whites, non-Hispanics blacks, and Hispanics in the USA. BMC Cancer. 2009; 9:129. [PubMed: 19400958]
14. Soto-Salgado M, Suárez E, Calo W, Cruz-Correa M, Figueroa-Vallés NR, Ortiz AP. Incidence and mortality rates for colorectal cancer in Puerto Rico and among Hispanics, non-Hispanics whites, and non-Hispanics blacks in the United States, 1998–2002. Cancer. 2009; 115(21):5126–7.
15. Fritz, G.; Percy, C.; Jack, A.; Sobin, LH.; Parkin, MD. International Classification of Diseases for Oncology. 3. Geneva: World Health Organization; 2000.
16. Surveillance Epidemiology and End Results (SEER). [accessed on February 1, 2012] SEER Behavior Recode for Analysis. [Internet] Available from: http://seer.cancer.gov/behavrecode/
17. [accessed August 4, 2011] NAACR Uniform Data Standard committee Subcommittee on methodological problems in measuring cancer in Hispanics. Final Report of Atlanta Symposium. [Internet] Available from: http://www.naacr.org/LinkClick.aspx?fileticket=pzDOtUUz9k0%3D&tabid=95&mid=477
18. Waller, LA.; Gotway, CA. Applied spatial statistics for public health data. Hoboken: John Wiley & Sons, Inc; 2004.
19. United States Census Bureau. [accessed on December 5, 2012] Percent Changes. [Internet] Available from: http://www.census.gov/acs/www/Downloads/data_documentation/Accuracy/PercChg.pdf
20. Tiwari RC, Clegg LX, Zou Z. Efficient interval estimation for age-adjusted cancer rates. Stat Methods Med Res. 2006; 15(6):547–69. [PubMed: 17260923]
21. Fleiss, J.; Levin, B.; Park, M. Fleiss, Levin & Park. Statistical methods for rates and proportions. 3. Hoboken: John Wiley & Sons, Inc; 2003.
22. Pickens A, Orringer MB. Geographical distribution and racial disparity in esophageal cancer. Ann Thorac Surg. 2003; 76(4):S1367–9. [PubMed: 14530066]
23. Castellsagué X, Muñoz N, De Stefani E, Victora CG, Castelletto R, Rolón PA, et al. Independent and joint effects of tobacco smoking and alcohol drinking on the risk of esophageal cancer in men and women. Int J Cancer. 1999; 82(5):657–64. [PubMed: 10417762]
24. Pelucchi, C.; Gallus, S.; Garavello, W.; Bosetti, C.; La Vecchia, C. Cancer Risk Associated With Alcohol and Tobacco Use: Focus On Upper Aero-Digestive Tract and Liver [Internet]. Bethesda, MD: National Institute on Alcohol Abuse and Alcoholism of the National Institutes of Health; Available from: http://www.nlm.nih.gov/bsd/uniform_requirements.html
25. Toh Y, Oki E, Ohgaki K, Sakamoto Y, Ito S, Egashira A, et al. Alcohol drinking, cigarette smoking, and the development of squamous cell carcinoma of the esophagus: molecular mechanisms of carcinogenesis. Int J Clin Oncol. 2010; 15(2):135–44. [PubMed: 20224883]
26. Morita M, Kumashiro R, Kubo N, Nakashima Y, Yoshida R, Yoshinaga K, et al. Alcohol drinking, cigarette smoking, and the development of squamous cell carcinoma of the esophagus: epidemiology, clinical findings, and prevention. Int J Clin Oncol. 2010; 15(2):126–34. [PubMed: 20224884]
27. Behavioral Risk Factor Surveillance System Survey (BRFSS). [accessed on June 30, 2011] Prevalence and Trends Data. [Internet] Available from: http://apps.nccd.cdc.gov/BRFSS/

28. Sakata K, Hoshiyama Y, Morioka S, Hashimoto T, Takeshita T, Tamakoshi A, et al. Smoking, alcohol drinking and esophageal cancer: findings from the JACC study. J Epidemiol. 2005; 15(Suppl II):S212–S219. [PubMed: 16127236]
29. Hajizadeh B, Rashidkhani B, Rad AH, Moasheri SM, Saboori H. Dietary patterns and risk of oesophageal squamous cell carcinoma: a case-control study. Public Nutr. 2010; 13(7):1107–12.
30. De Stefani E, Boffetta P, Deneo-Pellegrini H, Ronco AL, Correa P, Mendilaharsu M. The role of vegetable and fruit consumption in the aetiology of squamous cell carcinoma of the oesophagus: a case-control study in Uruguay. Int J Cancer. 2005; 116(1):130–5. [PubMed: 15756680]
31. CDC Morbidity and Mortality Weekly Report. State-specific trends in fruits and vegetables consumption among adults – United States 2000-2009. 2010; 59(35):1125–1130.
32. Jeon J, Luebeck EG, Moolgavkar SH. Age effects and temporal trends in adenocarcinoma of the esophagus and gastric cardia (United States). Cancer Causes Control. 2006; 17:971–81. [PubMed: 16841264]
33. Cossrow N, Falkner B. Race/ethnic issues in obesity and obesity-related comorbidities. J Clin Endocrinol Metab. 2004; 89(6):2590–4. [PubMed: 15181028]
34. Whiteman DC, Sadeghi S, Pandeya N, Smithers BM, Gotley DC, Bain CJ, et al. Combined effects of obesity, acid reflux and smoking on the risk of adenocarcinomas of the oesophagus. Gut. 2008; 57:173–80. [PubMed: 17932103]
35. Pérez CM, Guzmán M, Ortiz AP, Estrella M, Valle Y, Pérez N, et al. Prevalence of the metabolic syndrome in San Juan, Puerto Rico. Ethn Dis. 2008; 18(4):434–441. [PubMed: 19157247]
36. Lagergren J. Influence of obesity on the risk of esophageal disorders. Nat Rev Gastroenterol Hepatol. 2011; 8(6):340–347. [PubMed: 21643038]
37. Fernandez JR, Willig A, Jones A, Shriver MD, Albu J, Allison DB. Genetic admixture is associated with visceral adipose tissue in Puerto Rican women. Int J Body Com. 2006; 4(3):137–143.
38. Wajchenberg BL. Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. Endocr Rev. 2000; 21(6):697–738. [PubMed: 11133069]
39. Eisen GM, Sandler RS, Murray S, Gottfried M. The relationship between gastroesophageal reflux disease and its complications with Barrett's esophagus. Am J Gastroenterol. 1997; 92:27–31. [PubMed: 8995932]
40. American Society for Gastrointestinal Endoscopy. [accessed on June 30, 2011] GERD, Barrett's Esophagus and the Risk for Esophageal Cancer. [Internet] Available from: http://www.asge.org/patients/patients.aspx?id=402
41. Ricci F, Marmorato R, Pagán PC, Cangiano JL, López J, Soto-Salgado M. 2010 Internal medicine clinical research symposium integrating clinical research into medical practice: abstracts. P R Health Sci J. 2010; 29(4):413–414.
42. Altman KW, Stephens RM, Lyttle CS, Weiss KB. Changing impact of gastroesophageal reflux in medical and otolaryngology practice. Laryngoscope. 2005; 115:1145–1153. [PubMed: 15995499]
43. Abrams JA, Fields S, Lightdale CJ, Neugut AI. Racial and ethnic disparities in the prevalence of Barrett's esophagus among patients who undergo upper endoscopy. Clin Gastroenterol Hepatol. 2008; 6(1):30–4. [PubMed: 18063419]
44. Corley DA, Kubo A, Levin TR, Block G, Habel L, Rumore G, et al. Race, ethnicity, sex and temporal differences in Barrett's oesophagus diagnosis: a large community-based study, 1994–2006. Gut. 2009; 58(2):182–8. [PubMed: 18978173]
45. Stewart SL, Swallen KC, Glaser SL, Horn-Ross PL, West DW. Comparison of methods for classifying Hispanic ethnicity in a population-based cancer registry. Am J Epidemiol. 1999; 149(11):1063–1071. [PubMed: 10355383]
46. Doll R, Peto R. The causes of cancer: quatitative estimates of avoidable risks of cancer in the United States today. J Natl Cancer Inst. 1981; 66(6):1191–1308. [PubMed: 7017215]
47. Berretta M, Lleshi A, Fisichella R, Berretta S, Basile F, Li Volti G, et al. The role of nutrition in the development of esophageal cancer: what do we know? Front Biosci (Elite Ed). 2012; 4:351–357. [PubMed: 22201877]

48. Jeurnink SM, Büchner FL, Bueno-de-Mesquita HB, Siersema PD, Boshuizen HC, Numans ME, et al. Variety in vegetable and fruit consumption and the risk of gastric and esophageal cancer in the European prospective investigation into cancer and nutrition. Int J Cancer. 201210.1002/ijc.27517

49. Hajizadeh B, Jessri M, Moasheri SM, Rad AH, Rashidkhani B. Fruits and vegetables consumption and esophageal squamous cell carcinoma: a case-control study. Nutr Cancer. 2011; 63(5):707–713. [PubMed: 21614725]

50. Steevens J, Schouten LJ, Goldbohm RA, van den Brandt PA. Vegetables and fruits consumption and risk of esophageal and gastric cancer subtypes in the Netherlands Cohort Study. Int J Cancer. 201; 129(11):2681–2693.10.1002/ijc.25928 [PubMed: 21960262]

51. Kubo A, Corley DA, Jensen CD, Kaur R. Dietary factors and the risks of oesophageal adenocarcinoma and Barrett's oesophagus. Nutr Res Rev. 2010; 23(2):230–246. [PubMed: 20624335]

Table 1

Age-standardized incidence (per 100,000) of the EC histological types, by racial/ethnic group, sex, and study period.

EC Histology	Racial/ethnic group	Age-standard incidence						Change (%) 2001–2005 vs. 1992–1996	
		1992–1996		1997–2000		2001–2005			
		Men	Women	Men	Women	Men	Women	Men	Women
All EC	PR	49.7	17.4	42.6	15.1	33.3	9.6	−33.0*	−44.8*
	NHW	26.9	8.8	29.6	8.8	31.8	8.9	18.2*	1.1
	NHB	48.7	13.8	33.6	13.6	32.0	11.0	−34.3*	−20.3*
	USH	23.1	5.6	27.3	6.3	23.8	5.1	3.0	−8.9
AC	PR	4.9	1.5	6.9	1.5	6.4	1.5	30.6	0.0
	NHW	14.8	2.4	19.0	2.8	21.4	3.5	44.6*	45.8*
	NHB	3.2	0.7	3.1	0.7	2.8	1.3	−12.5	85.7
	USH	8.5	1.5	13.7	2.6	12.8	2.1	50.6	40.0
SCC	PR	33.0	11.7	25.2	9.4	18.4	6.0	−44.2*	−48.7*
	NHW	9.1	5.1	8.2	4.5	7.2	4.0	−20.9*	−21.6*
	NHB	40.7	11.5	26.6	10.2	25.3	8.1	−37.8*	−29.6*
	USH	12.0	3.0	10.1	3.1	8.5	1.8	−29.2*	−40.0*

*The significant percentage of change was determined by the construction of the 95% CIs using the formulas from the US Census Bureau.[19] If zero was not included in this interval, significant changes were declared with *P*-values < 0.05.

Table 2

Age-specific incidence (per 100,000) for all EC by sex and racial/ethnic group during 2001–2005.

	Incidence				RR (95% CI)		
	PR	**NHW**	**USH**	**NHB**	**PR vs. NHW[a]**	**PR vs. USH[a]**	**PR vs. NHB[a]**
Men							
50–59 years	14.11	15.33	7.90	21.38	0.92 (0.76–1.12)	1.79 (1.36–2.35)*	0.66 (0.52–0.83)*
60–69 years	31.97	35.30	20.90	48.27	0.91 (0.78–1.06)	1.53 (1.22–1.92)*	0.66 (0.54–0.81)*
70–79 years	45.04	57.26	37.24	57.17	0.79 (0.67–0.93)*	1.21 (0.95–1.53)	0.79 (0.63–0.99)*
≥ 80 years	72.11	59.75	53.42	45.79	1.21 (1.00–1.45)	1.35 (1.01–1.80)*	1.57 (1.13–2.19)*
	SRR (95% CI)[b]				1.05 (0.93–1.17)[c]	1.40 (1.16–1.69)*	1.04 (0.88–1.23)[c]
Women							
50–59 years	1.03	2.60	0.81	6.73	0.40 (0.21–0.75)*	1.28 (0.53–3.07)	0.15 (0.08–0.30)*
60–69 years	5.59	7.36	3.08	13.78	0.76 (0.54–1.06)	1.82 (1.07–3.09)*	0.41 (0.28–0.60)*
70–79 years	14.00	15.54	5.74	22.23	0.90 (0.69–1.17)	2.44 (1.54–3.88)*	0.63 (0.45–0.88)*
≥ 80 years	25.75	20.07	13.88	17.57	1.28 (1.00–1.65)	1.86 (1.22–2.82)*	1.47 (0.99–2.17)
	SRR (95% CI)[b]				1.08 (0.89–1.30)[c]	1.88 (1.37–2.63)*	0.87 (0.68–1.10)[c]

[a] Reference group;

[b] Ratio of the two specified ASR(World) values;

[c] Age and racial/ethnic group interaction ($P < 0.05$);

* $P < 0.05$

Table 3

Age-specific incidence (per 100,000) for AC by sex and racial/ethnic group during 2001–2005

	Incidence				RR (95% CI)		
	PR	**NHW**	**USH**	**NHB**	**PR vs. NHW[a]**	**PR vs. USH[a]**	**PR vs. NHB[a]**
Men							
50–59 years	3.53	11.69	5.12	2.70	0.30 (0.21–0.44) *	0.69 (0.44–1.07)	1.31 (0.75–2.27)
60–69 years	5.27	24.45	9.66	3.51	0.22 (0.15–0.31) *	0.55 (0.35–0.85) *	1.50 (0.81–2.78)
70–79 years	7.88	38.60	17.51	7.68	0.20 (0.14–0.30) *	0.45 (0.29–0.71) *	1.03 (0.57–1.86)
≥ 80 years	14.10	38.55	29.77	2.86	0.37 (0.25–0.54) *	0.47 (0.29–0.78) *	4.93 (1.49–16.28) *
	SRR (95% CI)[b]				0.30 (0.23–0.38) *	0.50 (0.36–0.69) *	2.32 (1.51–3.71) *
Women							
50–59 years	0.41	1.08	0.08	0.61	0.38 (0.14–1.05)	5.11 (0.57–45.74)	0.68 (0.19–2.40)
60–69 years	1.58	2.78	1.17	1.47	0.57 (0.30–1.05)	1.35 (0.54–3.35)	1.07 (0.43–2.67)
70–79 years	2.19	4.63	1.61	1.69	0.47 (0.25–0.90) *	1.36 (0.52–3.58)	1.30 (0.47–3.57)
≥ 80 years	3.31	8.47	6.07	2.70	0.39 (0.20–0.76) *	0.55 (0.24–1.26)	1.22 (0.44–3.44)
	SRR (95% CI)[b]				0.43 (0.26–0.63) *	0.73 (0.40–1.36)	1.14 (0.60–2.22)

[a] Reference group;

[b] Ratio of the two specified ASR(World) values;

* $P < 0.05$

Table 4

Age-specific incidence (per 100,000) for SCC by sex and racial/ethnic group during 2001–2005

	Incidence				RR (95% CI)		
	PR	**NHW**	**USH**	**NHB**	**PR vs. NHW[a]**	**PR vs. USH[a]**	**PR vs. NHB[a]**
Men							
50–59 years	7.91	2.62	2.52	17.45	3.02 (2.26–4.03)*	3.14 (2.03–4.87)*	0.45 (0.34–0.61)*
60–69 years	21.94	8.14	9.49	40.53	2.69 (2.19–3.32)*	2.31 (1.68–3.18)*	0.54 (0.43–0.68)*
70–79 years	26.74	14.04	17.51	44.37	1.91 (1.52–2.39)*	1.53 (1.10–2.13)*	0.60 (0.46–0.80)*
≥ 80 years	36.33	13.86	16.79	34.34	2.62 (1.98–3.46)*	2.16 (1.34–3.50)*	1.06 (0.71–1.59)
	SRR (95% CI)[b]				2.56 (2.16–3.00)[c]*	2.16 (1.65–2.88)*	0.73 (0.60–0.89)[c]*
Women							
50–59 years	0.62	1.25	0.73	5.41	0.50 (0.22–1.14)	0.85 (0.30–2.39)	0.11 (0.05–0.27)*
60–69 years	3.01	3.84	1.61	11.21	0.79 (0.50–1.24)	1.87 (0.90–3.88)	0.27 (0.16–0.44)*
70–79 years	9.63	8.91	3.90	18.85	1.08 (0.79–1.48)	2.47 (1.41–4.32)*	0.51 (0.35–0.75)*
≥ 80 years	16.18	8.03	3.47	10.81	2.01 (1.45–2.79)*	4.66 (2.20–9.91)*	1.50 (0.91–2.46)
	SRR (95% CI)[b]				1.50 (1.16–1.89)[c]*	3.38 (2.18–5.54)[c]*	0.74 (0.56–0.99)[c]*

[a]Reference group;

[b]Ratios of the two specified ASR(World) values;

[c]Age and racial/ethnic group interaction ($P < 0.05$);

*$P < 0.05$